Pathophysiology of Sepsis and Shock in Dogs and Cats

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Sepsis represents a dysregulated host response to infection leading to life-threatening organ dysfunction. The progression from infection to sepsis to septic shock follows a continuum of increasing severity with distinct pathophysiological changes affecting multiple organ systems.

Cardiovascular Manifestations

Early sepsis presents with vasodilation, reduced systemic vascular resistance, and increased cardiac output (hyperdynamic phase). Nitric oxide production increases, causing widespread vasodilation and vascular permeability. Distributive shock results from this maldistribution of blood flow.

As sepsis progresses, myocardial depression develops from circulating myocardial depressant factors. Cardiac dysfunction manifests as biventricular dilation, reduced ejection fraction, and impaired contractility. The late hypodynamic phase features decreased cardiac output, hypotension, and poor tissue perfusion.

Microcirculatory dysfunction occurs independently of macrocirculatory changes, with heterogeneous blood flow, microthrombi formation, and endothelial cell damage contributing to tissue hypoxia despite seemingly adequate global oxygen delivery.

Metabolic Derangements

Cellular metabolism shifts dramatically during sepsis. Initially, hypermetabolism with increased oxygen consumption and glucose utilization occurs. Later, mitochondrial dysfunction leads to impaired oxidative phosphorylation and ATP depletion despite adequate oxygen availability (cytopathic hypoxia).

Lactate production increases from both tissue hypoperfusion and altered cellular metabolism. Persistent hyperlactatemia correlates with increased mortality in dogs and cats with sepsis.

Coagulation Abnormalities

Sepsis activates coagulation pathways while simultaneously suppressing anticoagulant mechanisms. Tissue factor expression increases, triggering thrombin formation. Concurrently, protein C, protein S, and antithrombin activities decrease, and fibrinolysis becomes impaired.

These changes create a procoagulant state leading to disseminated intravascular coagulation (DIC) with paradoxical microthrombi formation and bleeding tendencies. Microthrombi further compromise microcirculation and contribute to multiple organ dysfunction.

Organ Dysfunction

Kidney injury manifests through multiple mechanisms including hypoperfusion, direct inflammatory damage, and microvascular dysfunction. Acute tubular necrosis frequently develops, leading to oliguria and azotemia.

Respiratory dysfunction presents as acute respiratory distress syndrome (ARDS) characterized by increased vascular permeability, neutrophil infiltration, and surfactant dysfunction. Ventilation-perfusion mismatching and decreased lung compliance result in progressive hypoxemia.

Gastrointestinal dysfunction includes bacterial translocation from intestinal barrier breakdown, contributing to endotoxemia. Hepatic dysfunction manifests as hyperbilirubinemia and impaired protein synthesis.

Brain dysfunction (septic encephalopathy) occurs from blood-brain barrier dysfunction, microglial activation, and neurotransmitter alterations, resulting in altered mentation.

Anesthetic Considerations for Septic Patients

Preoperative Assessment

Thorough evaluation of cardiovascular, respiratory, renal, and hepatic systems is essential. Stabilization before anesthesia should include fluid resuscitation, vasopressor support if needed, and correction of electrolyte and acid-base abnormalities.

Laboratory workup should include complete blood count, serum biochemistry, coagulation profile, blood lactate, and blood gas analysis. Point-of-care ultrasound can assess volume status and cardiac

function.

Anesthetic Management Strategies

Fluid Therapy: Crystalloid fluid therapy remains first-line, aiming to restore intravascular volume while avoiding fluid overload. In hypotensive patients unresponsive to fluids, vasopressors should be implemented early.

Drug Selection: Anesthetic induction should employ agents with minimal cardiovascular depression. Etomidate provides cardiovascular stability but may suppress adrenal function. Low-dose ketamine with benzodiazepines or propofol offers a suitable alternative with some inotropic effects.

For maintenance, volatile anesthetic requirements are often reduced in septic patients. Consider partial intravenous anesthesia with constant rate infusions of fentanyl, lidocaine, or ketamine to reduce inhalant concentrations.

Cardiovascular Support: Vasopressors may be necessary to maintain adequate perfusion pressure. Norepinephrine (0.1-0.5 μg/kg/min) is preferred for septic shock due to its predominant α1-adrenergic effects with modest β-adrenergic activity. For patients with myocardial dysfunction, dobutamine (2-10 μg/kg/min) can improve cardiac output.

Ventilation Strategy: Lung-protective ventilation with tidal volumes of 6-8 mL/kg and minimizes ventilator-induced lung injury. Recruitment maneuvers may improve oxygenation when indicated.

Temperature Management: Active warming prevents hypothermia, which exacerbates coagulopathy and increases anesthetic drug effects.

Risk Stratification and Monitoring

High-risk features include persistent hypotension, hyperlactatemia >4 mmol/L, thrombocytopenia, coagulopathy, and multiple organ dysfunction. Patients with these findings require intensive monitoring.

Monitoring should include continuous ECG, pulse oximetry, capnography, blood pressure (preferably direct arterial), central venous pressure when possible, temperature, and urine output. Serial blood lactate measurements help assess adequacy of tissue perfusion.

Goal-directed therapy targets include:

- Mean arterial pressure >65 mmHg
- Central venous oxygen saturation >70%
- Lactate clearance
- Adequate urine output (>0.5 mL/kg/hr)

Post-Anesthetic Care

Close monitoring should continue postoperatively with particular attention to cardiovascular support, ventilation, and organ function. Ongoing fluid therapy should be carefully titrated to avoid volume overload. Multimodal analgesia with opioids and regional techniques minimizes respiratory depression.

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